Fortnightly Review

Pressure sores

R K Vohra, C N McCollum

Pressure sores have defied the best attempts of medical and nursing staff since they were first described in Egyptian mummies.[1] They are defined as a localised area of tissue damage resulting either from direct pressure on the skin causing pressure ischaemia or from shearing forces causing mechanical stress to the tissues. The pathophysiology remains poorly understood.[2]

Pressure sores are painful, unsightly, difficult to treat, and costly. The estimated cost of caring for pressure sores is £150 million a year in the United Kingdom and more than $3 billion in the United States.[3,4] A full thickness sacral sore typically involves substantial expenditure in staff time, dressing materials, drugs, and special pressure relieving beds during hospital inpatient care over many months; the estimated total cost per patient is £26 000.[5] Also, there is the cost to patients in lost income, productivity, and independence, which the patients and their relatives increasingly attempt to recover from health authorities by litigation.[6,7]

Summary points

- Pressure sores are a financial burden to the NHS
- Morbidity and mortality with pressure sores are high
- Understanding aetiology will help concentrate resources on prevention
- Use of risk assessment scales may reduce the incidence of pressure sores by increasing awareness
- There is little evidence of the efficacy of topical dressings and applications
- Flap surgery is indicated when conservative treatment fails, pressure sores recur, and scars are unstable

Prevalence

The prevalence of pressure sores among hospital inpatients in the United Kingdom is 7-8%; it may be even higher in community medicine.[8,9] Elderly people are particularly susceptible, with 70% of all pressure sores occurring in patients aged over 70 years.[10] Younger patients usually have an underlying neurological disorder, and patients with cerebral palsy, multiple sclerosis, and spinal cord lesions are particularly susceptible: up to 85% of paraplegics develop a pressure sore.[11-13] Wheelchair users are also at a special risk, and around a quarter will develop a sore.[14]

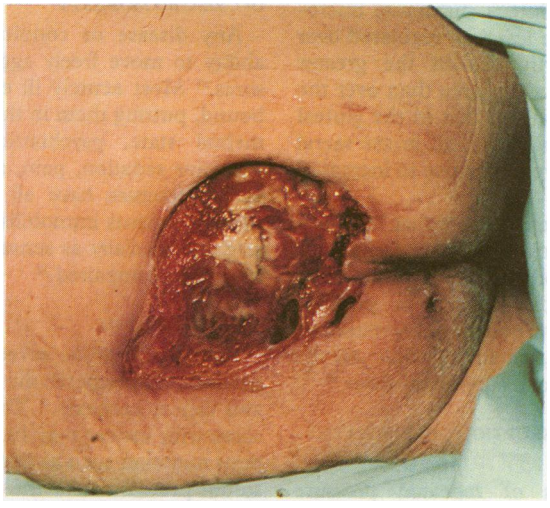

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FIG 1—*Stage IV sacral pressure sore showing muscle destruction*

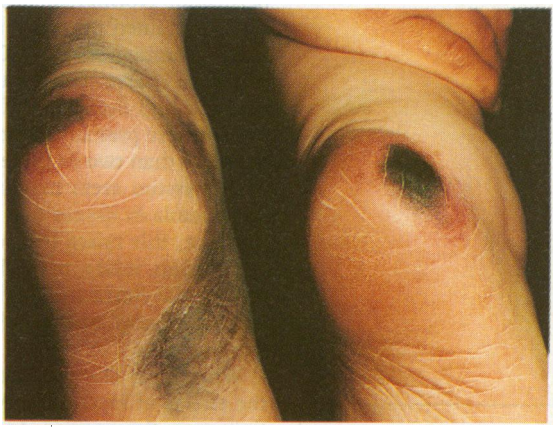

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FIG 2—*Stage III pressure sore on the heel*

Complications

Infection, dehydration, anaemia, electrolyte imbalance, and malnutrition often complicate pressure sores.[4,15] Infection may be manifested by generalised sepsis, osteomyelitis, or pyoarthrosis and carries a substantial mortality.[15-17] Pressure sores may be grossly underreported in official statistics based on death certificates—they were recorded as a cause of death in only 171 patients and mentioned in fewer than 2000 certificates throughout all of 1986.[18]

Anatomy

Pressure sores (figs 1, 2) occur most commonly on the lower half of the body, particularly over the sacrum (43%), greater trochanter (12%), heel (11%), ischial

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tuberosities (5%), and lateral malleolus (6%).[19] These are the bony prominences that support the weight of the body during lying, sitting, and standing. Several classification systems have been devised for pressure sores; the one recommended by the American National Pressure Ulcer Advisory Panel (box) has become accepted as a standard.[20] This classification not only helps the clinician to select the right treatment but may also predict the prognosis.

Classification of pressure sores[20]

Stage I	Non-blanchable erythema of the intact skin. This is a red or violaceous area that does not blanch when pressed, indicating that blood has escaped from capillaries into the interstitial tissues
Stage II	Partial thickness skin loss. The skin surface is broken resulting in an abrasion or shallow crater
Stage III	Full thickness skin loss and extension into subcutaneous fat but not through underlying fascia
Stage IV	Extensive destruction involving damage to muscle, bone, or tendon

Causes: local factors

PRESSURE

Pressure sores are caused by compression of, or repeated trauma to, tissue covering a bony prominence. In the supine position the pressure over the buttocks is around 9·4 kPa whereas that on the ischial tuberosities in a sitting position can reach 40 kPa.[21] In children the highest pressures were recorded in the occipital area; sacral pressures were higher only in older and larger children.[22] The greater the pressure, the less time is needed for tissue necrosis due to impaired capillary perfusion. Normal fit people adjust their posture to avoid sustained pressure, and measurements over the bony prominences of 980 seated subjects showed a tolerance curve with an inverse relation between pressure and duration.[14] Tissue damage is thought to occur where pressures more than 9·3 kPa are sustained for more than two or three hours.[23] The erythema seen over pressure areas may initially be a simple reactive hyperaemia in tissues that were formerly hypoxic from pressure; this raises the possibility that an ischaemia-reperfusion injury may also be involved and that a resulting accumulation of inflammatory mediators and leucocytes may sustain inflammation, induration, and hyperaemia.

When laser Doppler fluxmetry was used to evaluate the effects of local skin pressure on skin microcirculation over the sacrum and gluteus maximus, microvascular flow was found to be less well regulated over the sacrum. This may partly explain the greater prevalence of pressure sores in this area than over the gluteal region. The skin blood cell flux in the gluteal region shows a more stable pattern than in the sacral area, where the flux signal is very sensitive to increased pressures.[24]

Interface pressure is the only variable that has been routinely measured and shown to be of practical importance. Simple electropneumatic or fluid filled sensors may be constructed in sheets to produce pressure mapping systems.[14,25] These have been used to measure interface pressures, including that of the interface between the subject and support surfaces.[25]

CAPILLARY OCCLUSION AND DISRUPTION OF LYMPHATIC DRAINAGE

The physiological effects of repeated pressure include capillary occlusion and disruption of lymphatic drainage. The effect of external loading on the skin microcirculation has been assessed with radioisotope clearance, photoplethysmography, transcutaneous oxygen tension measurements, and laser Doppler flowmetry.[14] There is no evidence that a critical closing pressure analogous to the capillary blood pressure can be identified, although higher pressures produce greater disruption of the microcirculation.[14] The lymphatic drainage of the subcutaneous tissues is also impaired by pressure: at 8·0-9·3 kPa lymphatic flow (measured by technetium-99m labelled sulphur) ceases.[14]

SHEARING FORCE

Reichel reported that patients more often developed pressure sores in sacral tissues when the head of the bed was raised.[26] Shear is a result of movement generated by the patient, attendants, or gravity. The skin and superficial tissue adhere to the bedclothes and are pulled tightly over the deep fascia, stretching, angulating, and traumatising the underlying blood vessels, which leads to thrombosis. The subcutaneous fat lacks tensile strength and is particularly susceptible to damage by these shearing forces.[13]

INCREASED TEMPERATURE AND MOISTURE

Cushions and mattresses are designed to retain heat, warming those tissues closely applied to their surfaces and exacerbating the effects of ischaemia by increased metabolic rate. This effect may persist for over an hour after relief from pressure.[27] Increased temperature also induces sweating, which alters skin integrity through maceration and compromises the natural barrier to infection. Maceration of skin also follows contamination by urine or faeces, wound drainage, and food spillage.[21] For these reasons patients with incontinence of urine and faeces are at particular risk.

Causes: systemic factors

AGING

Elderly patients are more susceptible to pressure necrosis owing to changes in aging skin, loss of subcutaneous tissue, diminished pain perception, decreased cell mediated immunity, slowed wound healing, and the altered barrier properties of aged skin.[28] The important skin changes in aging include decreased proliferative activity in the epidermis, flattening of the dermal-epidermal junction, attenuated microvasculature, reduced local inflammatory response, sensory loss, and diminished elasticity. All these, except possibly the reduced local inflammatory response, greatly increase the effect of local pressure and shear stress in creating pressure necrosis.

DECREASED MOBILITY

Any disease or condition impairing the patient's ability to move freely aggravates the risk of pressure sores.[21] Most acutely ill elderly patients will be bedbound, putting them in the highest risk category. Poor mental state, psychiatric or neurological disease, excessive sedation, pain, and orthopaedic injury such as hip fracture have all been strongly implicated.[28] Elderly patients immobilised by stroke are particularly at risk, especially as sensation over the pressure areas may also be impaired.[15]

POOR NUTRITION

Nutrition may be severely compromised in elderly patients, hypermetabolic states, prolonged pyrexia, and cancer cachexia.[21] The increased risk of pressure sores with cancer may be related either to features of poor nutrition such as depleted lymphocyte count and serum proteins or to the direct effect of those factors that cause cachexia.[29] Even the relatively simple

Risk factors for pressure sores

Local risk factors:
- Pressure
- Capillary occlusion and disruption of lymphatic drainage
- Shearing force
- Increased temperature and moisture

Systemic risk factors:
- Aging
- Decreased mobility
- Poor nutrition
- Arterial disease and hypotension

measure of hypoalbuminaemia is significantly related to pressure sores.[15] Chronic inflammation and infection due to the pressure sore itself may aggravate hypoproteinaemia and anaemia, which are usually refractory to treatment with iron and red cell transfusion but can be treated with recombinant human erythropoietin.[30]

ARTERIAL DISEASE AND HYPOTENSION

Patients with arterial disease, especially occlusion of large proximal vessels such as the aorta or iliac arteries, are particularly likely to develop pressure sores, as relatively small local pressures will impair tissue perfusion when the arterial filling pressure is low. It is important that the adequacy of the arterial supply is measured by Doppler techniques whenever there are pressure sores on the feet or when the femoral pulse is weak or absent with sacral or trochanteric sores. In the operating theatre, time on the operating table, prolonged hypotension, and extracorporeal circulation are all related to pressure necrosis.[31]

In the same way, hypotension from any cause is important and is potentially a factor precipitating pressure sores in patients with spinal cord injury.[32] In a prospective study of pressure sore risk among institutionalised elderly patients, low systolic and diastolic pressures emerged as important risk factors.[33] Again, the pressure needed to occlude capillary blood flow in tissues over the bony prominences is lower in hypotension. This may also be shown by the correlation between a low resting skin blood cell flux on laser Doppler fluxmetry and low mean blood pressures.[34]

Risk scales

The Braden (fig 3) and Norton scales have been recommended by the Agency for Health Care Policy and Research for predicting the risk of pressure sores.[35] These scales quantify a range of risk factors by using ratings whose summative scores are the basis for risk of pressure sores.[36-41] Predictions are based on mobility, activity, level of consciousness or sensory perception, incontinence, nutrition, friction, and shear. Although simple clinical scales of this type have a great deal of appeal owing to their ease of use, additional factors such as diastolic blood pressure, temperature, dietary protein intake, age, and stroke should not be ignored.[15 42] To complicate matters further, a low level of

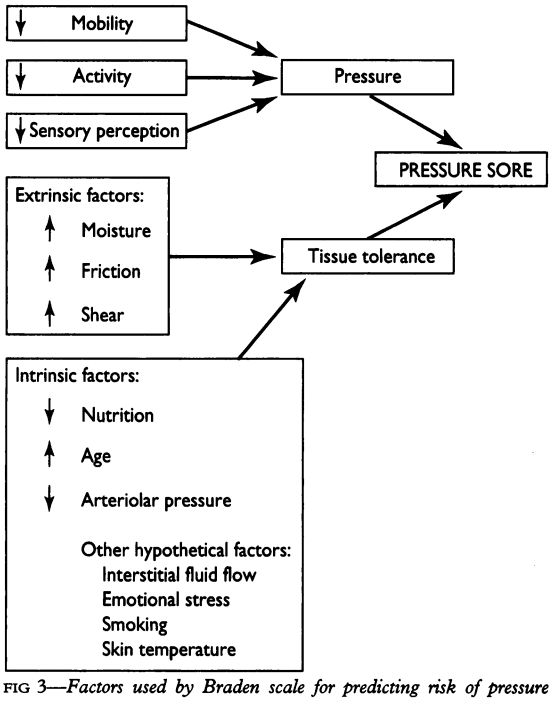

FIG 3—*Factors used by Braden scale for predicting risk of pressure sores*

education, unwillingness to practise standing, poor muscle tone, and frequent urinary infections all contribute to recurrence of pressure sores in patients after spinal injury.

Early diagnosis

Where sacral induration is detected, raised serum creatine phosphokinase concentrations may be helpful in distinguishing simple inflammation from impending tissue necrosis. Concentrations increase within two hours of release of pressure and remain raised for a week if necrosis has occurred.[43]

The susceptibility of high risk elderly patients to pressure sores can be predicted by recording the effect of pressure on skin microcirculation by using laser Doppler fluxmetry.[31] The recovery time after pressure relief is three times longer in patients who are at greatest risk of ulceration than in normal patients.[44]

Prevention

GENERAL MEASURES

Proper positioning, frequent weight shifts, lying prone at night, the elimination of shear and friction, care of body fluids, and frequent skin inspections are all standard nursing practices to prevent pressure sores.[45] Educational programmes may also help, reducing the incidence of pressure sores in elderly hospitalised patients by as much as 65%.[46] The real value in formally applying risk scales to all appropriate patients might be the increased awareness and focused nursing attention that would accompany the introduction of such a policy.

SPECIALISED BEDS, MATTRESSES, AND CUSHIONS

In an attempt to distribute pressure more evenly away from bony prominences, a variety of mattress overlays including foam, sheepskin, gel, and air products have become available. These may be classified by measuring interface pressure.[47] Foam mattresses produce higher local pressures than alternating air pressure mattresses in elderly patients and are more likely to predispose the patients to pressure sores.[48]

Of the pressure relieving beds, fluidising systems have been the most consistent at reducing pressure over the bony high points, but the low air loss bed also provides safe pressure relief.[49 50] In a comparison of alternating air, static air, and water mattress overlays on sacral and heel pressures in a surgical intensive care unit, mean pressures were significantly higher for the alternating air mattress than the other surfaces; they should therefore be avoided.[51] Further prospective studies are necessary before these beds become even more widely used for long term treatment of nursing home patients with severe pressure sores.

Wheelchair cushions rarely result in ischial pressure readings below the capillary pressure as the surface area bearing the upper body weight is very small; over 50% of the body weight is supported on 8% of the sitting area at or near the ischial tuberosities.[45] In patients with spinal cord injuries, lowest pressures at the ischial tuberosities were recorded with the airfilled cushions.[52] Functional electrical stimulation has been shown to prevent pressure sores in these paraplegic patients by inducing shape changes in the buttocks and improving blood flow.[53-55]

Treatment of established pressure sores

MEDICAL TREATMENT

The basic principles of medical treatment of established pressure sores are shown in the box. A wide range of topical dressings and applications (box) is

marketed, usually with little evidence of efficacy.[56] There is currently no justification for using the more expensive of these products. Carefully conducted clinical trials are urgently needed to evaluate which products should be used in this all too common and very debilitating condition.

Principles of medical treatment of established pressure sores

Improvement in general health and nutrition
Restoration of tissue perfusion by relief of pressure
Maintaining a clean wound
Preventing or treating infection
Stimulation of granulation tissue
Arterial reconstruction where necessary

Medical treatments for pressure sores

Topical agents
- Antibiotics: neomycin, gentamicin, metronidazole
- Sugar, honey, and other monosaccharides
- Benzoyl peroxide
- Heavy metal ions: gold, bismuth, zinc, titanium
- Absorbable gelatin sponge
- Enzymatic debridement: streptokinase-streptodornase, collagenase, trypsin
- Human fibroblast growth factor

Dressings
- Hydrocolloid occlusive dressing
- Polyurethane film dressing
- Moisture vapour permeable dressing

Physical agents
- Hydrotherapy, particularly whirlpool bath
- Hyperbaric oxygen
- Ultrasound
- Electrotherapy
- Carbon dioxide laser

Systemic agents
- Antibiotics
- Zinc sulphate
- Vitamin C
- Insulin

SURGICAL TREATMENT

Flap surgery is indicated in those patients who fail to respond adequately to conservative treatment, who have recurrent pressure sores and unstable scars, and who no longer have adequate padding due to the ravages of previous sores.[57-63] An audit of pressure sores treated in a regional plastic surgery unit in Scotland from 1971 to 1990 showed a dramatic increase in flap repairs in the past 10 years, with a concomitant reduction in direct closure and skin grafting.[64] Although the initial results with muscle flaps are good, recurrence of pressure necrosis is depressingly common.[65,66] Forty three per cent of cutaneous and 33% of musculocutaneous flaps recurred over two to 12 years.[66] As lack of sensation in the flap may contribute to this high recurrence rate, neurosensory musculocutaneous flaps have been tried.[67,68]

Despite the numerous advances of modern medicine, pressure sores continue to challenge nursing and medical staff. They are a cause of substantial avoidable suffering and are a financial burden to the NHS. It is important that we better understand their causes in order to concentrate resources on their prevention. Increased awareness among nursing and medical staff, encouraged by the use of risk assessment scales, is likely to reduce their incidence. In the absence of carefully conducted clinical studies, the benefit of a variety of available medical treatments is doubtful.

Options for surgery

Debridement and abscess drainage
Direct closure
Split thickness skin grafts
Myocutaneous flaps
Neurosensory myocutaneous flaps

1 Thomson-Rowling J. Pathological change in mummies. *Proc R Soc Med* 1961;54:409-15.
2 Chapman EJ, Chapman R. Treatment of pressure sores: the state of the art. In: Tienery EJ, ed. *Clinical nursing practice.* Edinburgh: Churchill Livingstone, 1986.
3 Scales JT, Lowthian PT, Poole AG, Ludman WR. "Vaperm" patient support system: a new general purpose hospital mattress. *Lancet* 1982;ii:1150-2.
4 White GW, Mathews RM, Fawcett SB. Reducing risk of pressure sores: effects of watch prompts and alarm avoidance on wheelchair push-ups. *J Appl Behav Analysis* 1989;22:287-95.
5 Hibbs P. Pressure sores: a system of prevention. *Nursing Mirror* 1982;155: 1311-3.
6 Staas WE Jr, Cioschi M. Pressure sores—a multifaceted approach to prevention and treatment. *West J Med* 1991;154:539-44.
7 Robertson JC. £100 000 damages for a pressure sore. *Care, Science and Practice* 1987;5:2.
8 Dealey C. The size of the pressure sore problem in a teaching hospital. *J Adv Nurs* 1991;16:663-70.
9 Hawthorn P, Nyquist R. Pressure sore survey. *Nursing Times* 1987;83:45.
10 Young JB, Dobrzanski S. Pressure sores: epidemiology and current management concepts. *Drugs and Ageing* 1992;2:42-57.
11 Barbenel JC, Jordan MM, Nicol SM, Clark MO. Incidence of pressure sores in the Greater Glasgow Health Board area. *Lancet* 1977;ii:548-50.
12 Bliss MR. Prevention and management of pressure sores. *Update* 1988;36: 2258-68.
13 Reuler IB, Coonery TJ. The pressure sore: pathophysiology and principles of management. *Ann Intern Med* 1981;94:661-6.
14 Barbenel JC. Pressure management. *Prosthetics and Orthotics International* 1991;15:225-31.
15 Berlowitz DR, Wilking SVB. Risk factors for pressure sores: a comparison of cross-sectional and cohort-derived data. *J Am Geriatr Soc* 1989;37:1043-50.
16 Michocki RJ, Lamy PP. The problem of pressure sores in a nursing home population: statistical data. *J Am Geriatr Soc* 1976;24:323-8.
17 Allman RM, Laprade CA, Noel LB, Walker JN, Moorer CA, Dear MR, *et al.* Pressure sores among hospitalized patients. *Ann Intern Med* 1986;105: 337-42.
18 Davies K, Strickland J, Lawrence V, Duvcan A, Rowe J. The hidden mortality for pressure sores. *Journal of Tissue Viability* 1991;1:18.
19 Peterson NC. The development of pressure sores during hospitalization. In: Kenedi RM, Cowden JM, Scales JT, eds. *Bedsore biomechanics.* London: Macmillan, 1976:219-24.
20 National Pressure Ulcer Advisory Panel. Pressure ulcers prevalence, cost and risk assessment: consensus development conference statement. *Decubitus* 1989;2:24.
21 Low AW. Prevention of pressure sores in patients with cancer. *Oncology Nursing Forum* 1990;17:179-84.
22 Solis I, Krouskop T, Trainer N, Marburger R. Supine interface pressure in children. *Arch Phys Med Rehabil* 1988;69:524-6.
23 Kosiak M. Etiology of decubitus ulcers. *Arch Phys Med Rehabil* 1961;42: 19-29.
24 Schubert V, Fagrell B. Local skin pressure and its effects on skin microcirculation as evaluated by laser-Doppler fluxmetry. *Clinical Physiology* 1989;9:535-45.
25 Barbenel JC, Sockalingham S. A device for measuring soft tissue interface pressure. *J Biomed Eng* 1990;12:519-20.
26 Reichel SM. Shearing force as a factor in decubitus ulcers in paraplegics. *JAMA* 1958;166:762-3.
27 Finestone HM, Levine SP, Carlson GA, Chizinsky KA, Kett RL. Erythema and skin temperature following continuous sitting in spinal cord injured individuals. *J Rehabil Res Develop* 1991;28:27-32.
28 Levine JM, Simpson M, McDonald RJ. Pressure sores: a plan for primary care prevention. *Geriatrics* 1989;44:75-90.
29 Waltman NL, Bergstrom N, Armstrong N, Norvell K, Braden B. Nutritional status, pressure sores and mortality in elderly patients with cancer. *Oncology Nursing Forum* 1991;18:867-73.
30 Turba RM, Lewis VL, Green D. Pressure sore anaemia: response to erythropoietin. *Arch Phys Med Rehabil* 1992;73:498-500.
31 Kemp MG, Keithley JK, Smith DW, Morreale B. Factors that contribute to pressure sores in surgical patients. *Research in Nursing and Health* 1990;13: 293-301.
32 Mawson AR, Biundo DJ Jr, Neville P, Linares HA, Winchester Y, Lopez A. Risk factors for early occurring pressure sores following spinal cord injury. *Am J Phys Med Rehabil* 1988;67:123-7.
33 Bergstrom N, Braden B. A prospective study of pressure sore risk among institutionalized elderly. *J Am Geriatr Soc* 1992;40:747-58.
34 Schubert V. Hypotension as a risk factor for the development of pressure sores in elderly subjects. *Age Ageing* 1991;20:255-61.
35 Agency for Health Care Policy and Research. *Pressure ulcers in adults: prediction and prevention. Clinical practice guideline No 3.* Silver Springs, MD: AHCPR, 1992. (Publ No 92-0047.)
36 Norton D, McLaren R, Exton-Smith AN. *An investigation of geriatric nursing problems in hospitals.* London: Corporation for the Care of Old People, 1962.
37 Braden BJ, Bergstrom N. Clinical utility of the Braden scale for predicting pressure sore risk. *Decubitus* 1989;2:44-51.
38 Goldstone LA, Goldstone J. The Norton score: an early warning of pressure sores. *J Adv Nurs* 1982;7:419-26.
39 Goldstone LA, Roberts BV. A preliminary discriminant function analysis of elderly orthopaedic patients who will and will not contract a pressure sore. *Int J Nurs Stud* 1980;17:17-23.
40 Bergstrom N, Demuth PJ, Braden BJ. A clinical trial of the Braden scale for predicting pressure risk. *Nurs Clin North Am* 1987;22:417-28.
41 Bergstrom N, Braden BJ, Laguzza A, Holman V. The Braden scale for predicting pressure sore risk. *Nurs Res* 1987;36:205-10.

42 Vidal J, Sarrias M. An analysis of the diverse factors concerned with the development of pressure sores in spinal cord injured patients. *Paraplegia* 1991;29:261-7.
43 Hagisawa S, Ferguson-Pell MW, Palmieri VR, Cochran GVB. Pressure sores: a biochemical test for early detection of tissue damage. *Arch Phys Med Rehabil* 1988;69:668-71.
44 Meijer JH, Schut GL, Ribbe MW, Goovaerts HG, Nieuwenhuys R, Reulen JPH, *et al.* Method for the measurement of susceptibility to decubitus ulcer formation. *Med Biol Eng Comput* 1989;27:502-6.
45 Staas WE, Cioschi HM. Pressure sores: a multifaceted approach to prevention and treatment. *West J Med* 1991;154:539-44.
46 Moody BL, Fanale JE, Thompson M, Vaillancourt D, Symonds G, Bonasoro C. Impact of staff education on pressure sore development in elderly hospitalized patients. *Arch Intern Med* 1988;148:2241-3.
47 Clark M, Rowland LB. Preventing pressure sores: matching patient and mattress using interface pressure measurements. *Decubitus* 1989;2:34-9.
48 Clark M, Rowlands LB. Comparison of contact pressures measured at the sacrum of young and elderly subjects. *J Biomed Eng* 1989;11:197-9.
49 Ryan DW, Byrne P. A study of contact pressure points in specialized beds. *Clin Phys Physiol Meas* 1989;10:331-5.
50 Wild D. Body pressures and bed surfaces. *Nursing Standard* 1991;5:23-7.
51 Sideranko S, Quinn A, Burns K, Froman RD. Effects of position and mattress overlay on sacral and heel pressures in a clinical population. *Res Nurs Health* 1992;15:245-51.
52 Bar CA. Evaluation of cushions using dynamic pressure measurement. *Prosthet Orthot Int* 1991;15:232-40.
53 Ferguson ACB, Keating JF, Delargy MAS, Andrews BJ. Reduction of seating pressure using FES in patients with spinal cord injury. A preliminary report. *Paraplegia* 1992;30:474-8.
54 Levine SP, Kett RL, Cederna PS, Brooks SV. Electric muscle stimulation for pressure sore prevention: tissue shape variation. *Arch Phys Med Rehabil* 1990;71:210-5.
55 Levine SP, Kett RL, Gross MD, Wilson BA, Cederna PS, Juni JE. Blood flow in the gluteus maximus of seated individuals during electrical muscle stimulation. *Arch Phys Med Rehabil* 1990;71:682-6.
56 Knight AL. Medical management of pressure sores. *J Fam Pract* 1988;27:95-100.
57 Colen SR. Pressure sores. In: McCarthjy JG, ed. *Plastic surgery.* Vol 6. *The trunk and lower extremity.* Philadelphia: Saunders, 1990.
58 Paletta CE, Freedman B, Shehadi SI. The VY tensor fasciae latae musculocutaneous flap. *Plast Reconstr Surg* 1989;83:852-7.
59 Kroll SS, Hamilton S. Multiple and repetitive uses of the extended hamstring V-Y myocutaneous flap. *Plast Reconstr Surg* 1989;84:296-302.
60 Rojviroj S, Mahaisavariya B, Sirichativapee W, Suibnugarn C. Vastus lateralis myocutaneous flap: the treatment for trochanteric pressure sores in paraplegic patient. *J Med Asoc Thai* 1989;72:629-31.
61 Pena MM, Drew CS, Smith SJ, Given KS. The inferiority based rectus abdominis myocutaneous flap for reconstruction of recurrent pressure sores. *Plast Reconstr Surg* 1992;89:90-5.
62 Rawat SS, Mathurt BS. Transverse lumbar flap for sacral bed sores. *Plast Reconstr Surg* 1991;88:154-8.
63 Kuhn W, Luscher NJ, de Roche R, Krupp S, Zach GA. The neurosensory muscolocutaneous tensor fasciae latae flap: long term results. *Paraplegia* 1992;30:396-400,
64 McGregor JC, Hoogberrgen MM. Audit of pressure sores treated in a regional plastic surgery unit (1971-1990). *J R Coll Surg Edinb* 1991;36:399-401.
65 Disa JJ, Carlton JM, Goldberg NH. Efficacy of operative cure in pressure sore patients. *Plast Reconstr Surg* 1992;89:272-8.
66 Relander M, Palmer B. Recurrence of surgically treated pressure sores. *Scand J Plast Reconstr Surg* 1988;22:89-92.
67 Luscher NJ, de Roche R, Krupp S, Kuhn W, Zach GA. The sensory tensor fasciae latae flap: a 9-year follow-up. *Ann Plast Surg* 1991;26:306-11.
68 Leasavoy MA, Dubrow TJ, Korn HN, Cedars MG, Castro DJ. "Sensible" flap coverage of pressure sores in patients with meningomyelocele. *Plast Reconstr Surg* 1990;85:390-4.

Lesson of the Week

Crush syndrome following unconsciousness: need for urgent orthopaedic referral

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Patients who have been unconscious may develop crush syndrome, which requires urgent orthopaedic referral

The acute compartment syndrome occurs when increased pressure within osteofascial compartments results in local muscle ischaemia. If left untreated it may lead to muscle necrosis and contractures. The systemic manifestations of this—the crush syndrome—are the results of haemodynamic and metabolic disburbances and acute renal failure.[1] Failure to appreciate the importance of muscle necrosis as the underlying problem in the crush syndrome may have disastrous consequences.

The crush syndrome was originally described during the London Blitz in civilians who had been buried beneath the debris of destroyed houses.[2] Nowadays, victims are typically encountered in war zones, in mining disasters, after earthquakes, and in industrial or road traffic accidents.[3] The syndrome may also develop after isolated compression of arms or legs by the victim's own body—for example, during unconsciousness after a drug overdose.[1] Such patients often delay seeking medical attention or have other more apparent complications that need urgent attention. The local signs of muscle compression and necrosis may therefore initially be overlooked. We describe 11 such cases referred to our unit over 51 months.

Case reports

During April 1989 to July 1993, 11 patients were admitted to this infirmary with the crush syndrome secondary to a drug overdose. They were all referred for orthopaedic assessment after a considerable delay—mean 35 hours (table). Nine patients were men. The mean age was 31 years (range 20-54 years). As all the patients had been unconscious for an unknown length of time before presentation, the delay from time of injury until medical attention was given could not always be assessed. Four patients were unconscious on admission. Ten patients had taken an overdose of sedatives or painkillers, and one patient had carbon monoxide poisoning. During the period of unconsciousness their torso had compressed one or

Details of 11 patients with crush syndrome secondary to drug overdose 1989-93

Case No	Sex	Age (years)	Limb affected	Symptoms on admission	Time between admission and surgery (h)	Dialysis	Surgical treatment	Outcome	
								Functional	Renal
1	M	20	Upper arm	Yes	29	Yes	Fasciotomy with excision of necrotic muscles	Loss of flexion of elbow	Normal
2	M	24	Upper arm	Yes	24	Yes	Fasciotomy with excision of necrotic muscles	Stiff shoulder	Normal
3	M	35	Calf	NR	>62	Yes	Below knee amputation	—	Failure
4	M	54	Both calves	Yes	17	Yes	Bilateral above knee amputation	—	Failure
5	F	43	Calf	Yes	21	Yes	Through knee amputation	Died	
6	M	23	Forearm	Yes	30	No	Fasciotomy	Contractures	Normal
7	M	25	Calf	?		No	Through knee amputation	—	Normal
8	F	28	Forearm	Yes	51	No	Fasciotomy	Good	Normal
9	M	38	Forearm	Yes	22	No	Fasciotomy	Died	
10	M	24	Thigh	Yes	53	Yes	Fasciotomy	Died	
11	M	29	Forearm	Yes	43	Yes	Fasciotomy with excision of necrotic muscles	Loss of digital flexors	Normal

NR=not recorded.

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